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FATS AND FATTY ACIDS IN NUTRITION

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Fats have both a caloric and a noncaloric function in human nutrition. In the American diet they supply approximately 33 percent of the total per capita caloric intake, and perform a number of vital functions in the body which are unrelated to their action as energy bearing materials (1). These functions involve the "essential" unsaturated fatty acids which are present to a varying degree in the phospholipids and triglycerides of all plant and animal tissue. In addition, natural fats contain small amounts of tocopherols, carotenoids and sterols. However, the biological activity of these compounds are only indirectly related to those of the fatty acids.

Less than one-half of the total caloric fat intake is supplied as "visible" or prepared fats; the remainder is supplied by the "invisible" fat in meat, eggs, dairy products, cereals, nuts, fruits, and vegetables. The consumption of prepared fats in the form of butter, margarine, lard, shortening, and edible oils has varied between 43 to 46 pounds per capita, or a total of 5.3 to 6.5 billion pounds per year, for the last 20 years (2). It must be pointed out, however, that these fats have also been largely consumed as invisible fats in bakery products and in the preparation of food for table use.

The level of fat intake is obviously not only a question of food habits and national taste, but is also related to the standard of living and food availability. An optimum diet which is based on an adequate consumption of meat, eggs, and dairy products would naturally contain more fat than one based largely on cereals and vegetables. During World War II, fats supplied approximately 40 percent of the total caloric intake of the American soldier (3) in contrast to only 3 percent of the total caloric intake of the Oriental soldier (4) whose ration was based largely on cereals and vegetables.

The ready availability of prepared fats has been an important factor in the American diet. Three sources of secondary products from primary industries have contributed to this availability, the first, butter from the dairy industry, the second, lard and tallow from the meat packing industry, and the third, cottonseed oil from the cotton industry. Cottonseed oil was at first blended with tallow and marketed as a substitute lard. However, after the introduction of the catalytic hydrogenation process in 1910, this oil as well as other vegetable oils could be converted into a plastic or solid fat and was marketed as hydrogenated vegetable fat (5).

Chemical and Physical Properties of Triglycerides

No chemical distinction exists between plastic fats and edible oils; both contain glycerol esterified with three moles of fatty acids. If the glycerol is esterified with more than two unsaturated fatty acids, i.e. oleic or linoleic acid the resulting triglyceride is a liquid or an "oil" at room temperature. If, on the other hand, glycerol is esterified with only long chained saturated fatty acids or only one mole of oleic and two moles of palmitic or stearic acid, the resulting triglyceride is a solid or "fat" at room temperature.

Natural fats and oils have been found to contain mixtures of triglycerides which are uniquely characteristic of a specific fat (6). Lard and beef fat contain 5 to 15 percent trisaturated glycerides while cottonseed and soybean oils do not contain any trisaturated glycerides (7). The plastic fats or shortenings actually contain 15-35 percent of solid phase; the remainder is in a liquid phase incorporated into the solid phase to improve shortening performance and to keep the melting point of the mixture of triglycerides below body temperature. Vegetable oils are therefore hydrogenated in order to convert linolenic and linoleic to oleic and stearic acid so as to produce enough of the solid phase to convert the oil into a plastic fat at room temperature. The triglycerides in this solid phase have melting points well above body temperature, but the presence of the liquid phase acts as a solvent so that the plastic fats are liquids at 37°C. or 98.6°F.

Although no chemical distinction exists between plastic fats and edible oils, the latter should not be confused with mineral oil which is composed of hydrocarbons of no biological value. Because of its non-caloric value, mineral oil has been suggested as a replacement for edible oils in salad dressing in low fat diets (8). The use of mineral oil for this purpose is not recommended (9) as it interferes with the absorption of vitamin A and other desirable nutrients; furthermore, its use in prepared foods is banned in interstate commerce.

The shortening and the creaming value of plastic fats is dependent on their ability to lubricate and weaken the structure of wheat gluten and to hold air respectively. For flaky tender pastries and pies, a fat of high shortening and low creaming value should be incorporated in thin layers without excessive mixing so that the baked products break easily along the lines in which the fat was distributed. For soft fine texture cakes a fat of high creaming value should be used in order to incorporate a maximum amount of air, moisture and sugar by thorough mixing or creaming. The creaming value of fats can be enhanced by mixing them with 5 to 20 percent mono and diglycerides; the latter are prepared commercially by heating fats or fatty acids with glycerol under vacuum. The creaming value can also be en-

hanced by a new process (2, 10) which involves the rearrangement or interchange of the fatty acids in the triglycerides. The specific glycerides responsible for an undesirable crystal habit are broken up during this interchange and triglycerides with smaller crystals and more desirable creaming value are produced.

Although mono and diglycerides are not normally present in natural fats (11), proof of their innocuousness as a food ingredient has been partly based on the fact that they are found in the digestive system. According to Frazer the natural fats are partially hydrolyzed to mono and diglycerides in the intestinal tract and together with the bile salts form a highly effective emulsifying system (12). This system facilitates the entrance of approximately one-half of the ingested fat into the lymph; the rest is presumed to go directly to the liver via the portal blood. The stomach also plays an important role in fat digestion as it sends the chyme into the intestine at a rate in which it can be utilized (13). Conversely, the effect of fat on the emptying rate of the stomach contributes to the satiety value of food.

Oral Fat Emulsion

Fat emulsions, prepared by emulsifying an oil with mono and diglycerides, phospholipids, or synthetic emulsifiers have been used as caloric supplements whenever it was not possible to use all or part of the gastro-intestinal tract (14). When part of the tract was still available as in fractures of the lower jaw bone, oral emulsions proved successful (15). The nitrogen deficit brought about by a calorically inadequate diet could be corrected by the supplementary administration of an oral fat preparation containing 40 percent peanut oil and 10 percent dextrose. Furthermore, the absorption of orally fed corn oil was unaffected by the presence of purified soybean phosphatide or Tween 80 in amounts greatly in excess over the amount needed to make excellent emulsions (16). When none of the gastro-intestinal tract was available the intravenous administration of fat emulsions has been resorted to. However, technical difficulties have been encountered in this technic as such emulsions must be sterilized, not "break" after sterilization, and must not be pyrogenic. Phospholipids have been the best source of such emulsion stabilizers although impure preparations have caused histopathological lesions (14).

The Fate of Absorbed Fat

The fat absorbed through the blood and lymph is either deposited in the various tissues or used immediately as a source of energy. In the blood, fat appears to be transported in large part in the form of tiny droplets termed

"chylomicrons." The fatty acids are also present as components of phospholipids, cholesterol esters, and lipoproteins. Normal human blood plasma contains from 245 to 470 mg fatty acids, 120 to 350 mg phospholipids, and 110 to 310 mg cholesterol per 100 ml of plasma (17). A portion of the dietary fat which enters the blood via the lymph may first reach tissue other than the liver and may be deposited in the adipose tissue in the form of depot fats. Nevertheless, the liver appears to represent a major site of fat transformation.

The fats are in a continuous state of flux with the fatty acids moving from the fat depots to the liver and vice versa. Therefore, even though a wide variety of fats is consumed the mixed fatty acid composition of the body fat is fairly characteristic of the species (6) unless large amounts of highly unsaturated fatty acids are consumed. As the plastic hydrogenated fats which contribute a major share of the dietary fat are low in highly unsaturated fatty acids, deposition of substantial amounts of these acids would be unlikely.

The human body contains approximately 20 percent of ether extractable or "lipid" material (18). The amount of fat deposited is dependent on the tissue. The adipose tissue, heart, and skin, contain the most fat or 71.6, 16.6, and 14.2 percent respectively, and the lungs and spleen the least fat or 1.3 and 1.2 percent respectively. Striated muscle contains 6.6 percent, liver 3.1 percent, and the kidney 7.2 percent fat. The adipose tissue fat contains approximately 4 percent (C_{14}) myristic, 25 percent (C_{16}) palmitic, 7 percent (C_{18}) stearic, 6 percent (C_{16}) palmitoleic, 46 percent (C_{18}) oleic, and 2 percent of fatty acids which are shorter than 14 or longer than 18 carbon atoms in chain length (19). These fatty acids may be classified the "non-essential fatty acids" as they can all be synthesized in the body from non-fat precursors. In addition, the body fat contains approximately 9 percent (C_{18}) linoleic and 1 percent (C_{20}) arachidonic acid which contain two and four double bonds or points of unsaturation respectively. These two fatty acids have been classified the "essential fatty acids" as linoleic acid can not be synthesized in the body and serves as an essential precursor for the synthesis of arachidonic acid.

The Metabolism of Fats and Fatty Acids

There is more combustible material in a gram of fat than in a gram of protein or carbohydrate as fat is richer in carbon and hydrogen. If burned in a bomb calorimeter, approximately 9300, 5600, and 4200 calories would be obtained from a gram of fat, protein, and carbohydrate respectively (20). In the body this energy is also released by oxidation to carbon dioxide and

water but much more gradually through a complex system of enzymes operating at 98.6°F. A part of the mechanisms in which the enzymes involved in the metabolic oxidation of fats operate has recently become known (21).

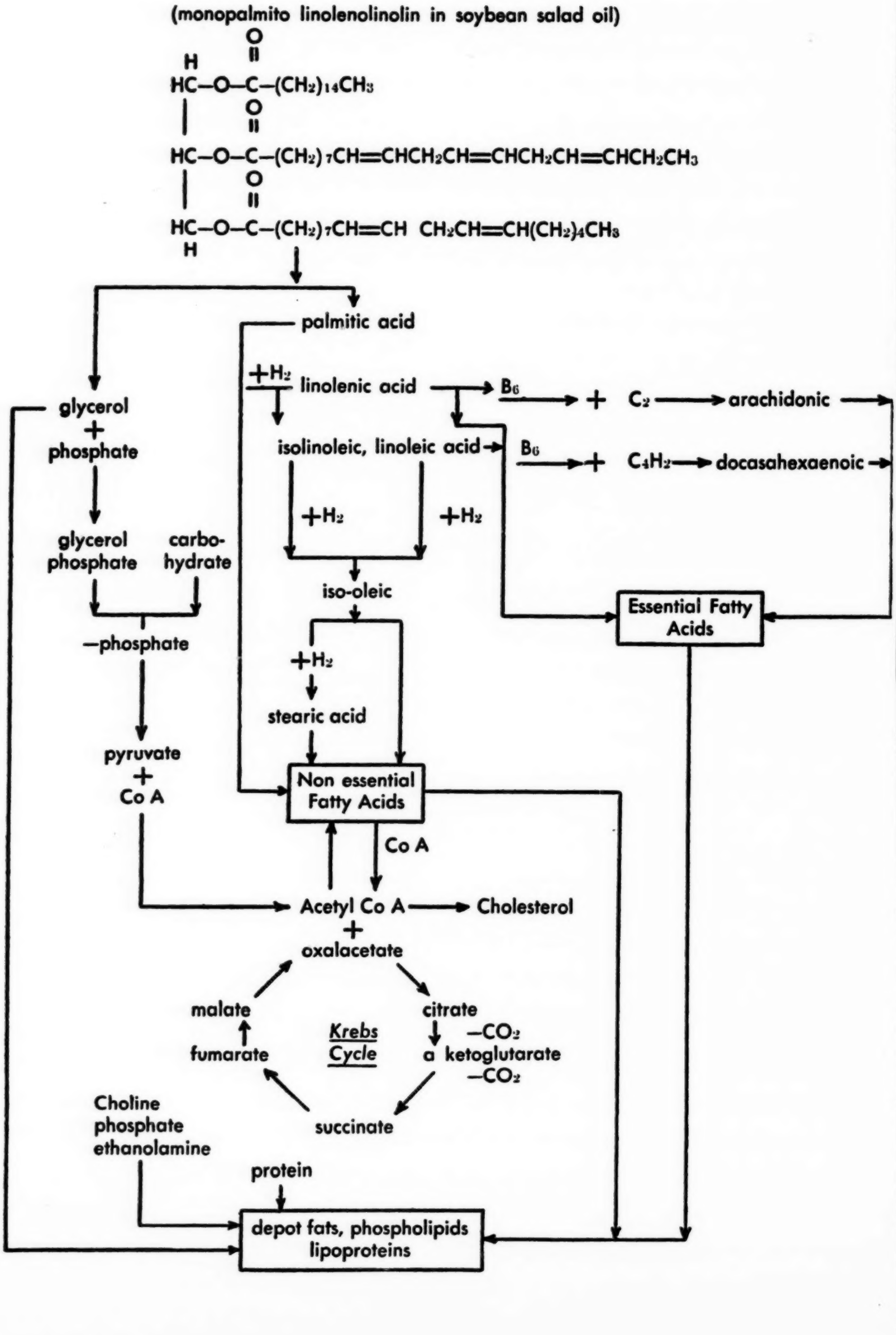
It has been shown that the normal oxidation of fats is dependent on the presence of one or more of the breakdown products of carbohydrate metabolism. These breakdown products are formed in what is known as the Krebs citric acid cycle, the system in which carbohydrates are metabolized. It is believed that before breakdown, fats are hydrolyzed to glycerol and free fatty acids. The glycerol combines with phosphate and glycero-phosphate so formed (22) oxidized to pyruvic acid which is also the key breakdown product of glucose to enter the citric acid cycle. In the presence of the appropriate enzyme system and the energy-rich adenosine triphosphate (ATP) pyruvic acid can act as an acetylating agent and can convert coenzyme A into "acetyl coenzyme A" (23). Coenzyme A or "Co A" is a derivative of the vitamin pantothenic acid and contains in addition, phosphate, adenylic acid, and mercaptoethanolamine ($NH_2CH_2CH_2SH$). The acetyl group is believed to be linked to the coenzyme by means of a thiol ester linkage.

The free fatty acids are normally completely oxidized to carbon dioxide and water through the citric acid cycle (24). They are first esterified with Co A in the presence of ATP, a two-carbon fragment splits off to give acetyl Co A and the Co A derivative of the next lower fatty acid. Thus a long-chain fatty acid can be completely degraded to C_2 fragments without the intermediate formation of free short-chain fatty acid. The acetyl Co A combines with oxaloacetate to form citrate (25). The oxaloacetate is subsequently regenerated through the citric acid cycle with the formation of two molecules of carbon dioxide corresponding to the two carbon atoms of the acetate entering the cycle (26).

The acetyl Co A which is not metabolized to carbon dioxide and water can add to the metabolic pool of acetyl Co A made available through the metabolism of carbohydrates and some of the amino acids. This pool does not actually exist under normal conditions, as acetyl Co A is immediately used to build up new fatty acids and new fats, non-essential amino acids, sterols and other lipid material. The evidence to date suggests that the non-essential unsaturated fatty acids palmitoleic and oleic acid are synthesized from the corresponding saturated fatty acids palmitic and stearic acid with the aid of tissue dehydrogenases (27).

Therefore, any one of the non-essential fatty acids can be consumed as dietary fat or synthesized from acetyl Co A. Furthermore, the consumption of a low fat diet will not prevent fatty acid synthesis unless the total caloric intake is cut drastically enough to bring about a subnormal synthesis of acetyl

Metabolism of a Hypothetical Mixed Triglyceride in Dietary Fat



Co A. Such action would seem undesirable as it might interfere with the synthesis of other vital biological components.

The mechanism involved in the synthesis of fatty acids through a multiple condensation of acetyl Co A is believed to involve a reversal of those or similar reactions involved in the degradation of fatty acids (28, 29, 30). The free fatty acids do not detectably accumulate, but are immediately transformed into glycerol esters. The glycerol esters formed from carbohydrates *in vivo* would be indistinguishable from the glycerol esters in animal or vegetable shortenings except in one respect. If it were possible to isolate the newly synthesized triglycerides and to determine their glyceride composition, differences between the glyceride composition of this fat and animal and vegetable shortenings would be noted. Unfortunately, the triglyceride composition of human fat has not as yet been determined and such comparisons can not be made at this time.

The energy requirements of the body are normally met by metabolizing fat and carbohydrate together. If for any reason carbohydrate metabolism is subnormal, correspondingly more fat has to be metabolized. Under these conditions there is a deficiency of carbohydrate metabolites and a tendency for acetyl Co A to accumulate for lack of oxaloacetate with which to combine to yield citrate (20). Two carbon units then tend to accumulate and pairs to combine to yield acetoacetate, from which β-hydroxybutyric acid and acetone the other two members of the "ketone bodies" can form. Thus ketone bodies accumulate when the diet contains disproportionately large amounts of fat, when the glycogen reserves of the liver have been exhausted as in starvation, or when the liver's power to store and metabolize glycogen is seriously impaired as in diabetes.

The Relationship of Atherosclerosis to Cholesterol

Human atherosclerosis, which is the most prevalent type of arteriosclerosis, is responsible for 25 percent of all deaths in the United States (31). Clinical observations have shown that a high correlation exists between obesity and atherosclerosis (32) and clinical tests such as cholesterol and lipoprotein or Sf 10-20 molecule determinations of blood serum (33) also seem to indicate a correlation between fats and atherosclerosis. Furthermore, the feeding of large amounts of cholesterol to herbivorous animals such as rabbits and chickens leads to the deposition of cholesterol in the arterial walls and to atherosclerosis. Therefore, attempts (34, 35, 36) to establish a relationship between human atherosclerosis and the ingestion of cholesterol and fat may have a seemingly logical basis. However, this conclusion has been questioned by many investigators (31, 32, 37) and does not agree with the biochemical data.

These data indicate that cholesterol can be synthesized readily from vegetable fat, carbohydrate, and protein *in vivo*. Acetyl Co A as well as acetoacetic acid and isovaleric acid, a product of the metabolic degradation of the amino acid leucine, can be utilized for cholesterol synthesis (25). Although the liver is the major site of synthesis, other tissues are also capable of this process (38). In fact, even arterial tissue has been shown to synthesize cholesterol (39).

The total caloric intake is therefore more important to cholesterol synthesis than a low-fat, low-cholesterol diet. The synthesis of either fat or cholesterol would not be restricted by consuming a low-fat, low-cholesterol diet unless the caloric intake is drastically curtailed. A low-fat, low-protein diet such as the rice-fruit diet fed at a level below the minimum daily requirement does lead to a lower blood cholesterol level but it also leads to a negative nitrogen balance, and is not well enough tolerated to be used continuously (32). Furthermore, it has been pointed out that high cholesterol and lipoprotein values of blood are not abnormal and that diabetic children as well as adults can develop arteriosclerosis (31). It has, therefore, been suggested that arteriosclerosis is a metabolic disease and that a more intensive study of endocrine factors would seem in order.

In addition, two recent studies serve to emphasize the lack of correlation between atherosclerosis and the ingestion of dietary fat. One, rats which had been raised on a fat-free diet contained twice as much liver cholesterol but had lower blood cholesterol levels than those on a 15 percent fat diet (40). Two, a diet deficient in the amino acids methionine and cystine caused atherosclerosis in monkeys (41). Therefore a balanced diet adequate in complete proteins, enough fat to insure optimum protein utilization (42) and adequate amounts of other essential nutrients, combined with moderate exercise (43), would seem a better method of controlling obesity and its possible consequences than a low-fat, low-protein diet.

The Essential Fatty Acids

The 9 percent of "essential" linoleic acid, which was shown to be present in human fat must originate in dietary fat. Most of the experimental proof for the need of dietary sources of essential fatty acids has been obtained with rats as an experimental animal, as it would be difficult to feed a fat-deficient diet to men long enough to produce fat deficiency symptoms. However, human infants on a fat-free diet have developed an eczema which was readily cured by feeding fat (44). This type of eczema was accompanied by a lowering in serum linoleic and arachidonic acid values. On the basis of this evidence and on the observation that rats kept on a fat-deficient diet develop a characteristic dryness and "scurfiness" on the dorsal surface of the

hind feet, the essential fatty acids are believed to be necessary for the maintenance of normal skin tissue (45).

The one percent of arachidonic acid present in human fat is either synthesized from linoleic acid *in vivo* or consumed as a component of animal fats such as lard. Vegetable oils or hydrogenated vegetable oils do not contain arachidonic acid. However, when an adequate amount of pyridoxine is fed with a source of linoleic acid, such as vegetable oils, arachidonic acid is synthesized *in vivo*. Another highly unsaturated fatty acid, linolenic acid present in linseed and soybean oil, also seems to function as an "essenital" fatty acid provided enough pyridoxine is fed (46).

In the presence of pyridoxine and to a lesser degree certain antioxidants (47) linolenic acid may be converted to a more highly unsaturated six double bond or docosahexaenoic acid (48). It is therefore possible to divide the highly unsaturated fatty acids into two groups: (a) the two double bond linoleic which can be converted to the four double bond arachidonic and (b) the three double bond linolenic which may be converted to a six double bond docosahexaenoic acid. Linolenic, however, is not so efficient as linoleic or arachidonic acid in stimulating growth when fed to rats which had been kept on a fat-deficient diet; it can not alleviate acrodynia or acute dermal symptoms (47), and can not prevent abnormal reproduction in rats kept on a low-fat diet (49).

The Dietary Need for Linoleic Acid for Normal Reproduction

Female rats which had been raised to maturity on a diet deficient in fat, but adequately supplied with other dietary factors and bred with normal males, could not give birth to normal living young unless 5 percent corn oil was supplied in the diet (50). Labor began at term in the "fat-deficient" animals; however, parturition was accompanied by excessive hemorrhage. Parturition was not completed for two or three days, while the animals lost weight and became extremely weak and anemic. Approximately 14 percent of the females died before giving birth to their young; 80 percent of the latter were born dead and all of the young which were born alive died within 24 hours after birth. Total fat analyses of representative female rats and their newly-born young indicated that the fat "deficient" animals, that is the animals which had been kept on the fat-free diet, were deficient in arachidonic acid and not deficient in fat as such.

The Disposition of the Highly Unsaturated Fatty Acids in Various Tissues

The heart, liver, and brain contain higher concentrations of linoleic and

arachidonic acid than the adipose, skin, and skeletal muscle tissue (51). The heart tissue fatty acids from normal rats contain a higher concentration of highly unsaturated fatty acids and are more responsive to dietary changes than any other tissue studied thus far. Furthermore, a higher concentration of linoleic and arachidonic acid is found in the phospholipid than in the neutral fat or triglyceride fractions.

The American diet normally contains small amounts of iso-oleic and iso-linoleic acid which are formed from linoleic and linolenic acid during the hydrogenation of vegetable oils. An iso-oleic acid, vaccenic acid, has also been found in butterfat and has been erroneously classified as a growth promoting factor (52, 53). All of the natural isomers of linoleic and linolenic acid seem to be metabolized without difficulty (54). Moreover, the small amount of linolenic acid which is present in the fat of nuts, vegetables, and salad oil made from soybean oil seems to be metabolized under normal conditions by man, rat, and chicken. However, if more than 5 percent of a rich source of linolenic acid such as linseed oil is fed to chickens, as much as 25 percent of linolenic acid is found in the skin tissue fat (55).

The Relationship Between Autoxidized Fats and Nutrition

Although it is not known whether unsaturated fatty acids combine directly with oxygen *in vivo,* the ease with which they combine with oxygen *in vitro* to produce autoxidized or "rancid" fats has an important bearing on the nutritive value of fats. The autoxidized fats possess beany, grassy, painty, or fishy flavors which will lead to rejection of the food item in which these off-flavors appear. The rate of development of "rancid" odors and flavors in a particular food item depends on many variables, among which are its fat content, the degree of unsaturation of the fat, the processing and storage conditions, the presence of various chemicals or of lipoxidase (56) and the presence of antioxidants.

Tocopherol, the natural antioxidant in edible fats (57) is not destroyed during hydrogenation and thus contributes to the stability of hydrogenated vegetable shortenings. The stabilizing value of antioxidants is now being taken advantage of by the animal fats industry through the addition of government-approved antioxidants to lard and tallow. Antioxidants can not, however, counteract completely the deleterious effects of processing to which some foods are subjected during preparation for table use. On exposure to heat, autoxidation can begin and continue during the storage period. Although autoxidation is retarded at low temperature, it does go on even at freezer locker temperatures; in meats it is stimulated by the presence of the enzyme lipoxidase (56).

Mechanism Involved in the Autoxidation of Fats

The autoxidation of fats seems to involve at least five phases; the induction period, peroxide formation, peroxide decomposition, polymerization, and degradation. After an initial induction period, all four of the remaining phases occur simultaneously. However, the predominant phase at the moment a test sample is taken is dependent on the length of time the autoxidation has proceeded. For example, a sample taken during the second stage of autoxidation would contain predominantly peroxide oxygen and in addition small amounts of polymers and the products of degradation, i.e., aldehydes and ketones.

Commercially processed fats can develop off-flavors and odors without developing "rancidity" through peroxide formation. It is possible unintentionally to introduce polymers into the fresh oil or fat during processing by passing it through polymer-contaminated equipment (58). Furthermore, these "oxidative" polymers are not eliminated during the hydrogenation process (59) and can, on storage, break down to produce off-flavors in the hydrogenated shortening.

Although changes have not been apparent during the induction period, changes nevertheless do occur. It is known that antioxidants are oxidized during this time. As carotenoids can act as antioxidants, some of the natural fats, such as beef tallow, which owe their yellow-orange color to these pigments are bleached white. Moreover, peroxide oxygen can not be detected until all of the antioxidant is oxidized; the length of the induction period is governed by the effectiveness of the antioxidant and its concentration in the fat.

During the second or peroxide-accumulating phase, both the organoleptic and analytical characteristics change. The fat develops "rancid" odors and undesirable flavors which change in character as the fat becomes more oxidized. The analytical characteristics indicate that the peroxide oxygen adds momentarily at the double bond and then shifts to the "active" methylene group next to the double bond (60). If two or more double bonds are present, a simultaneous shift from a nonconjugated to a conjugated double bond system occurs. On further autoxidation, 1,4 addition of oxygen across the conjugated double bond system takes place and the oxidized monomers combine through carbon-to oxygen linkages to form dimers and trimers (61). If these oxidative polymers are isolated from the autoxidated fat and reautoxidized, they degrade to volatile carbonyl compounds (62). Acetaldehyde, propionaldehyde, n-hexanal, and 2-pentenal have been isolated from the oxidative polymers of autoxidized soybean oil (58).

It has been shown that oxidative polymers degrade to volatile carbonyl compounds under vacuum as well as under oxygen-free nitrogen (58).

Therefore, the packaging of food under vacuum or nitrogen in air-tight tin cans can only prevent the autoxidation of unsaturated triglycerides but not the decomposition of the oxidative polymers to volatile carbonyl compounds. Furthermore, most edible oils as well as other foods are subjected to variable storage periods before processing and thus are exposed to oxygen and possible autoxidation. Unfortunately, analytical tests which can be used to detect oxidative changes before peroxides are formed are still not available. Furthermore, even if peroxides are formed they may be destroyed during processing. For example, hydrogenation or toasting will remove possible peroxides from vegetable oil or cereals respectively, but can not remove oxidative polymers from either.

The Nutritive Value of Polymerized Fats

Two types of polymerized fat can readily be formed; one, a heat or thermal polymerized fat prepared by heating the fat under vacuum at a high temperature and two, an oxidative polymerized fat prepared by exposing fat to oxygen for a long period of time. If fats are heated in the presence of air both types of polymers probably form in the fat.

Heat polymerized fish oils have been used extensively in Norway as a substitute for soybean and olive oil in the packing of sardines and for an edible salad oil (63). On the other hand, the use of heat polymerized fish oils for edible purposes has been banned in some sections of Germany (64). Heat-treated linseed oil has been found to have less nutritional value than fresh linseed oil (65). However, as linseed oil is not used as a frying, baking or cooking fat, these results can not be used as a guide for judging the performance of the more stable hydrogenated vegetable fats under similar conditions. A highly rancid soybean oil, which undoubtedly contained oxidative polymers, supported almost normal growth when fed to rats at a 10 percent level on a 30 percent protein diet (66).

In Summary

From both a biological and an economic viewpoint it seems fortunate that the food habits of the American people call for approximately one-third of the caloric intake as fats. This amount of fat should provide for an adequate amount of "essential" fatty acids and an adequate amount of fat for the optimum use of protein. Furthermore, the total caloric intake and lack of exercise rather than the consumption of dietary fat leads to obesity and its consequences.

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Dr. Fred A. Kummerow obtained a B.S. degree in chemistry and an M.S. and Ph.D. degree in biochemistry at the University of Wisconsin. From 1943 to 1945 he worked under a grant from the Williams-Waterman fund for the combating of dietary diseases at Clemson College, Clemson, South Carolina. He left Clemson in 1945 for a position in the chemistry department at Kansas State College. At the latter institution he directed research on the mechanism involved in the autoxidation of unsaturated fatty acids *in vitro* and the metabolism of these acids *in vivo*. He has been at the University of Illinois since 1950 and is continuing his work on unsaturated fatty acids. He has been the author or co-author of 42 papers and is a member of the Associates, Food and Container Institute — for the Armed Forces and the journal committee of the American Oil Chemists' Society.

REVIEW OF SYMPOSIUM ON GERONTOLOGY

(Sponsored by the National Vitamin Foundation and the Johns Hopkins University School of Hygiene and Public Health)

On Tuesday, March 2, 1954, at the Biltmore Hotel in New York, a symposium was conducted to provide doctors and other scientists, particularly those in the field of nutrition, an opportunity to hear and evaluate some of the most recent clinical findings on the relation of nutrition and the aging process.

The following reports were presented at the meeting: The Present Status of Physiological and Biochemical Studies of Aging, by Nathan W. Shock; The Role of Comparative Physiology in the Study of Aging, by Dr. A. I. Lansing; Fat Metabolism—With Particular Reference to Problems of Aging, by Dr. H. J. Deuel, Jr.; Protein Metabolism—With Particular Reference to Problems of Aging, by Dr. C. S. Davidson; The Absorption of Vitamins and Enzymic Activities in Aging, by Dr. B. F. Chow; Some Observation on Blood and Urine Vitamin Levels in the Aged, by Dr. J. E. Kirk; Hemopoietic Factors with Reference to Aging, by Dr. F. H. Bethell; The Hormones and Metabolism, by Dr. C. N. H. Long; Endocrine Stress and Aging, by Dr. D. J. Ingle; and Special Nutrition Problems of the Aged, by Dr. H. A. Rafsky.

The papers that were presented indicated clearly that very little indeed is known about the fundamental biochemical and physiological transformations concerned in the aging process. The symposium did much however to delineate some of the important aspects of aging that require emphasis in research programs. It was interesting to hear the point of view expressed that aging begins when growth stops, which stated in another way indicates that as aging proceeds reactions of anabolism diminish and those of catabolism tend to increase.

Studies reported indicated that as age advances, the fat content of the body increases which is accompanied

by a gradual decrease in the water content. Although the general blood lipid pattern does not seem to be altered in a definite manner with aging, there does seem to be a significant increase in blood cholesterol levels with increasing age.

Evidence was also presented which demonstrates an increase in the percent of underweight people with advancing age and a decrease in lean body mass, or protein content with age. It was not completely clear whether or not this observation was influenced by the adverse longevity statistics of the obese and overweight. It was emphasized, that in general, food ingestion decreases with age, particularly protein intake. Much of this seems to be related to the psychological factors which affect adversely the aged person's inclination to prepare and eat a well balanced diet.

Old people apparently have a lowered ability to absorb vitamin B_{12}, and also a lower level of tissue saturation with respect to this vitamin. This may in part be explained by the observation that the "binding power" or intrinsic factor content of gastric juice may be reduced in the aged.

In a report on hematopoietic factors in the aged, it was noted that there is a tendency for a decline in hemoglobin and red blood cell levels in aging, but these declines were not great. In accord with an earlier reported observation it was reported that the level of intrinsic factor in gastric juice tends to decrease with age.